Individual variability in cardiac biomarker release after 30 min of high-intensity rowing in elite and amateur athletes

Alejandro Legaz-Arrese, Isaac López-Laval, Keith George, Juan José Puente-Lanzarote, Diego Moliner-Urdiales, Vicente Javier Ayala-Tajuelo, Carmen Mayolas-Pi, and Joaquín Reverter-Masia

Abstract:

This study had two objectives: (i) to examine individual variation in the pattern of cardiac troponin I (cTnI) and N-terminal pro-brain natriuretic peptide (NT-proBNP) release in response to high-intensity rowing exercise, and (ii) to establish whether individual heterogeneity in biomarker appearance was influenced by athletic status (elite vs. amateur). We examined cTnI and NT-proBNP in 18 elite and 14 amateur rowers before and 5 min, 1, 3, 6, 12, and 24 h after a 30-min maximal rowing test. Compared with pre-exercise levels, peak postexercise cTnI (pre: 0.014 ± 0.030 g·L–1; peak post: 0.058 ± 0.091 g·L–1; p = 0.000) and NT-proBNP (pre: 15 ± 11 ng·L–1; peak post: 31 ± 19 ng·L–1; p = 0.000) were elevated. Substantial individual heterogeneity in peak and time-course data was noted for cTnI. Peak cTnI exceeded the upper reference limit (URL) in 9 elite and 3 amateur rowers. No rower exceeded the URL for NT-proBNP. Elite rowers had higher baseline (0.019 ± 0.038 vs. 0.008 ± 0.015 g·L–1; p = 0.003) and peak postexercise cTnI (0.080 ± 0.115 vs. 0.030 ± 0.029 g·L–1; p = 0.022) than amateur rowers, but the change with exercise was similar between groups. There were no significant differences in baseline and peak postexercise NT-proBNP between groups. In summary, marked individuality in the cTnI response to a short but high-intensity rowing bout was observed. Athletic status did not seem to affect the change in cardiac biomarkers in response to high-intensity exercise.

Key words: exercise, cTnI, NT-proBNP, athletic status, rowing, elite athletes, amateur athletes.

Introduction

An increasing number of studies have described the elevation of cardiac troponin I (cTnI), a biomarker of cardiac cell necrosis, and N-terminal pro-brain natriuretic peptide (NT-proBNP), a biomarker of cardiac dysfunction, after prolonged and strenuous exercise (Scharhag et al. 2008; Shave et al. 2010a). The cardiac biomarker response to short-duration, high-intensity exercise is largely unknown, although some have suggested that within the endurance exercise domain, cTnI increases with exercise intensity (Legaz-Arrese et al. 2011; Serrano-Ostáriz et al. 2011). Shave et al. (2010b) are one of the few groups to have studied the cTnI response to short, high-intensity bouts of exercise. In spite of the limited volume of exercise (30 min all-out treadmill run), cTnI was elevated during recovery in 75% of athletes (Shave et al. 2010b).

Importantly, Shave et al. (2010b) observed that the appearance of cTnI during recovery was markedly heterogeneous and confirmed similar individuality of response in field-based studies of prolonged exercise (Shave et al. 2010a) as well as an observation from a meta-analysis (Shave et al. 2007). The percentage of individuals with postexercise cTnI or cardiac troponin T (cTnT) levels

above the upper reference limit (URL) has varied from 0% (Roth et al. 2007) to 100% (Middleton et al. 2008) in individual studies, but this may partially represent the "lottery" of a single postexercise blood test. It is important that in ongoing cardiac biomarker research, multiple postexercise blood draws occur to fully understand any heterogeneity in peak cTnI or NT-proBNP concentrations as well as recovery kinetics (Middleton et al. 2008).

The influence of exercise intensity on NT-proBNP release is less well understood. Within the endurance exercise domain, data suggest that NT-proBNP release may be influenced more by exercise duration than by exercise intensity (Serrano-Ostáriz et al. 2009), but studies involving shorter bouts of high-intensity exercise in well-trained athletes are limited.

Individual variability in biomarker response may be at least partially related to training or "athletic" status. It has been suggested that highly trained individuals have lower postexercise cTnI and NT-proBNP release (Mehta et al. 2012; Neilan et al. 2006). Indeed, the only 2 previous studies of elite athletes have shown normal postexercise cardiac biomarker levels (Bonetti et al. 1996; König et al. 2003). Conversely, we recently observed that a controlled endurance training intervention in untrained subjects resulted in higher pre- and postexercise values of cTnT with no changes in NT-proBNP (Legaz-Arrese et al. 2015). The influence of training level on cardiac biomarker release has not yet been evaluated in a controlled study in which groups that differ in terms of training or athletic status complete a similar (relative) highintensity exercise bout. Finally, it has been postulated that increases in both cTnI and NT-proBNP may be dependent on the respective resting values (Legaz Arrese et al. 2005; Serrano-Ostáriz et al. 2011), although this potential dependence has not been studied in different athlete groups.

Consequently, the purpose of the present study was to determine the cardiac biomarker response to a short-duration, highintensity bout of rowing, with specific emphasis on detailing individual responses across multiple assessment points during a 24-h recovery period. A secondary purpose was to determine the influence of athletic status on cTnI and NT-proBNP release by comparing 2 cohorts: amateur and elite rowers.

Material and methods

Participants

Thirty-two male rowers were recruited from a large rowing club in Spain through an open invitation to all of its members. Volunteers included elite rowers (n = 18) who had at least 3 years of competitive history at the national or international level (1 world champion, 1 under-23 world champion, 1 Olympic competitor, 2 Spanish champions, and 3 Spanish sub-champions) and were training ≥5 days per week and noncompetitive amateur rowers (n = 14) who trained ≤3 days per week. All rowers provided written informed consent. The study followed the ethical guidelines of the Declaration of Helsinki and was approved by the Research Ethics Committee of the Government of Aragón (CEICA, Spain).

Research design and protocols

All rowers attended a preliminary testing session 1 week before the main study was performed. At this initial testing, body height was measured to the nearest 0.1 cm (SECA 225, SECA, Hamburg, Germany). Body mass was determined to the nearest 0.05 kg (SECA 861, SECA, Hamburg, Germany). A questionnaire was used to obtain personal data, performance level, training history, and history of cardiac symptoms. Exclusion criteria were a significant personal or early family history of cardiovascular disease and (or) an abnormal electrocardiogram at baseline examination.

The rowers then performed a progressive incremental test to exhaustion on a rowing ergometer (Model C, Concept2, Morrisville, Vt., USA) so that the maximal heart rate (HR) could be determined (Polar Electro Oy, Kempele, Finland). Prior to the test, the rowerscompletedaself-paced5-minwarm-up(HR<130beats·min–1). The test began at a workload of 150 W (elite rowers) or 75 W (amateur rowers), and the workload increased by 50 W every 3 min until exhaustion. Strong verbal encouragement was provided to all participants.

On Saturday, 7 days after the progressive incremental test, all participants returned to the laboratory to complete the 30-min rowing test. All participants were fully habituated to the 30-min all-out rowing test protocol and were asked to abstain from strength training and strenuous exercise for 48 h before testing. Therefore, the last high-intensity training session was completed on Wednesday. All high-intensity testing sessions occurred at 1100 in a sports hall at a temperature of 18–21 °C and a relative humidity of 50%–60%. The rowers completed a self-paced 5-min warm-up (HR < 130 beats·min–1) followed by the 30-min all-out rowing test. Pairs of rowers competed side-by-side to mimic a regular competition and, again, strong verbal encouragement was provided. During the test, HR was recorded continuously with a Polar HR monitor (Polar Electro Oy, Kempele, Finland) and the data were downloaded using Polar Precision Performance software (version 3.0). The mean power output (W) and distance covered were recorded every 5 min from the rowing ergometer screen. Immediately after the test was completed, the participants rated the test for perceived exertion (RPE) (Borg and Kaijser 2006). Venous blood samples were taken before, immediately after (5 min), and 1, 3, 6, 12, and 24 h after exercise to assess serum cardiac-specific biomarkers.

Blood sampling and analysis

Blood samples were drawn by repetitive venipuncture from an antecubital vein and quickly centrifuged. The serum and plasma were drawn off and stored at –80 °C for later analysis. cTnI was analyzed from samples of EDTA (ethylenediaminetetraacetic acid) plasma with the Access AccuTnI assay (Beckman Coulter, Fullerton, Calif., USA). The imprecision profile of 839 duplicate samples showed 10% and 20% coefficient of variation (CV) values of 0.014 and 0.008 g·L–1, respectively. The URL for cTnI, defined as the 99th percentile for healthy participants, was 0.04 g·L–1 (Eggers et al. 2007). NT-proBNP was analyzed in the serum with an Elecsys proBNP electrochemiluminescent immunoassay on the Roche Elecsys 1010 (Roche Diagnostics, Lewes, UK) with an analytical range of 5 000 to 35 000 ng·L–1 and intra- and interassay imprecisions of 0.7%–1.6% and 5.3%–6.6%, respectively. The URL for NTproBNP was considered to be 125 ng·L–1 (Silver et al. 2004).

Statistical analysis

Statistical analyses were performed using the IBM Statistical Package for the Social Sciences (IBM SPSS Statistics, v. 20.0 for Windows). Cohort data are presented as the mean ± standard deviation unless otherwise stated. Kolmogorov–Smirnov tests were used to check for normal distribution, and data for cTnI and NT-proBNP were log-transformed prior to statistical testing. To measure the impact of sampling time (pre, 5 min, 1, 3, 6, 12, and 24 h postexercise) during recovery as well as athletic status (elite and amateur) on cTnI and NT-proBNP, we performed mixedmodel 2-way ANOVAs with post-hoc Bonferroni tests when appropriate. Differences between groups in the relative increase of cTnI or NT-proBNP were assessed by independent t test. A stepwise regression analysis was carried out to analyze the relationships between postexercise values of cTnI and NT-proBNP and several potential predictors (e.g., baseline cTnI and NT-proBNP concentrations, mean and maximum exercise HR, rowing performance, RPE). Differences and relationships were considered significant if $p < 0.05$.

Results

The characteristics of the elite and amateur rowers are shown in Table 1. The elite rowers had more years of training, greater weekly training frequency, and higher weekly training volume (all $p < 0.05$). Performance during the graded rowing test was

Table 1. Participant characteristics by athletic status.

	Age (y)	Weight (kg)	Height (cm)	Rowing training (y)	Rowing training frequency (sessions/wk)	Rowing training history volume (h/wk)
Elite rowers	21.0±4.1	77.9±6.0	181.4±6.0	8.2±5.4*	6.9±0.3*	22.1±6.6*
Amateur rowers	21.2±2.0	76.6±8.7	177.0±9.0	3.7±1.5	1.6±0.5	2.9±0.8

Note: Values are means ± standard deviation (elite rowers: $n = 18$; amat ur rowers: $n = 14$).
*Significant difference between elite and amateur rowers.

Table 2. Performance during the maximal 30-min rowing test.

	0–5 min power (W)	5–15 min power (W)	15–25 min power (W)	25–30 min power (W)	Mean power (W)	Percentage of maximum power (%)
Elite rowers	260±23*	254±22*	251±23*	286±27*	259±23*	88±3*
Amateur rowers	165±48	156±37	157±33	179±37	161±36	76±5

Note: Values are means ± standard deviation (elite rowers: $n = 18$; amateur rowers: $n = 14$).
*Significant difference between elite and amateur rowers. A similar pacing strategy was observed in both groups, with a significant increase in rowing performance in the last 5 min.

greater in the elite rowers (294 ± 18 W vs. 211 ± 44 W; $p = 0.000$), but maximum HR (elite: 196 ± 7 beats·min–1; amateur: 193 ± 9 beats·min–1; $p = 0.372$) and exercise duration (elite: 11.6 ± 1.7 min; amateur: 11.2 ± 2.6 min; $p = 0.544$) showed no significant statistical differences between groups.

Thirty-minute maximal rowing test

All of the subjects completed the 30-min maximal rowing test and every blood draw. Performance during the 30-min all-out test was substantially greater in the elite athletes (Table 2). Whilst mean HR was higher in the elite rowers (180 ± 7 beats·min–1) compared with the amateur rowers (171 ± 12 beats·min–1; $p = 0.023$), there was no difference in maximum HR (elite rowers: 195 ± 7 beats·min–1; amateur rowers: 188 ± 11 beats·min–1; $p = 0.061$) or RPE (elite rowers: 8.7 ± 0.5; amateur rowers: 8.6 ± 0.5; $p = 0.536$).

cTnI release

A significant main effect of sampling time was observed for cTnI, which was elevated at 3, 6, and 12 h postexercise compared with baseline (p = 0.000) (Table 3). All participants presented with an increase in cTnI postexercise. During recovery, the URL for cTnI was exceeded by 2 rowers at all measurement points, and another 10 rowers (8 elite and 2 amateur) exceeded the URL at sporadic time points (Fig. 1). The maximum postexercise cTnI was observed at 3 h in 11 individuals, 6 h in 19 individuals, and 12 h in 2 individuals. A significant main effect of athletic status was also observed: cTnI levels, including pre-exercise levels, were higher in elite rowers (amateur: 0.008 ± 0.015 g·L–1; elite: 0.019 ± 0.038 g·L–1; p = 0.003). There was no significant interaction of test time and athletic status with respect to cTnI (p = 0.311). In support of this, the maximal increase in cTnI (peak – baseline) was not significantly different between groups in either absolute terms (elite: 0.062 ± 0.083 g·L–1; amateur: 0.023 ± 0.021 g·L–1; p = 0.145) or relative terms (elite: 440% ± 382%; amateur: 1252% ± 1817%; p=0.398).Bothgroupsshowedsimilarvariability(CV)inpre-exercise (elite: 200%; amateur: 188%) and peak postexercise (elite: 144%; amateur: 98%) cTnI values. The stepwise regression analysis using maximum postexercise cTnI as the dependent variable and basal cTnI and mean exercise HR as independent variables yielded R2 = 0.810 (p = 0.000). Basal cTnI was identified as the best predictor of postexercise cTnI (R2 = 0.781, p = 0.000).

NT-proBNP release

There was a main effect of time: an increase in NT-proBNP from pre-exercise levels was observed at 5 min, 1, 3, 6, 12, and 24 h post-exercise (p = 0.001; Table 3). There was a rise in NT-proBNP postexercise in all subjects, but the URL was not exceeded by any subject (Fig. 2). The maximum postexercise NT-proBNP values were observed at 5 min in 10 individuals, 1 h in 4 individuals, 6 h in 3 individuals, 12 h in 4 individuals, and 24 h in 11 individuals.

There was no significant main effect of athletic status on NTproBNP levels, and there was no time by athletic status interaction effect. In support of the latter point, there was no difference between the elite and amateur rowers with respect to the maximum increase in NT-proBNP in absolute terms (14 ± 11 vs. 18 ± 13 ng·L–1, respectively; p = 0.470) or relative terms (115% ± 71% vs. 165% ± 213%, respectively; p = 0.536). After the stepwise regression analysis, the only variable significantly associated with the logarithm of maximum postexercise NT-proBNP values was the logarithm of basal NT-proBNP values (R2 = 0.697, p = 0.000). There was no correlation between changes in NT-proBNP and cTnI.

Discussion

The main findings of this study were as follows: (i) a single 30-min bout of “all-out” rowing exercise resulted in significant increases in cTnI and NT-proBNP in both elite and amateur rowers; (ii) there was significant individual heterogeneity in peak cTnI during recovery, with the URL for cTnI exceeded in 12 of 32 rowers; (iii) there was less individual variability in peak NT-proBNP, with no value above the URL; (iv) baseline and postexercise cTnI levels were higher in elite rowers; and (v) rowing-induced changes in cTnI and NT-proBNP were independent of athletic status.

Postexercise cTnI peak and kinetics in elite and amateur rowers

Our results in rowers extend the findings of Shave et al. (2010b), who demonstrated that cTnI is elevated following short-duration, high-intensity exercise (a 30-min high-intensity run) in non-elite athletes. An elevation in cTnI occurred in all participants despite the relatively short duration of

exercise and the limited exercise volume. There is some evidence to suggest that cTnI release during prolonged exercise is positively associated with exercise intensity (Fu et al. 2009; Serrano-Ostáriz et al. 2011; Shave et al. 2007). Whilst our study did not compare exercise intensities, it adds to the existing data that show that different types and intensities of exercise can stimulate an increase in circulating cTnI. According to the results of Shave et al. (2010b), cTnI release following shortduration intense exercise may be as common as cTnI release after prolonged exercise, and the current study supports this contention. This also underscores the necessity to complete blood draws during recovery (Middleton et al. 2008).

To our knowledge, this study is the first to demonstrate cTnI release with exercise in elite athletes, with values above the URL in some, but not all, participants. Previously, only 2 studies had evaluated cTnT release in elite athletes. Bonetti et al. (1996) analyzed 25 cyclists participating in the Giro d'Italia and detected cTnT in only 5 athletes; moreover, the cTnT values were below the cutoffs considered to be indicative of myocardial insult. Similarly, König et al. (2003) reported normal postexercise cTnT levels in 11 professional road cyclists. Both studies were constrained by limited blood sampling (pre- and postexercise design) and by less sensitive measurement equipment.

Although all participants experienced a rise in cTnI postexercise, the magnitude of peak postexercise cTnI was variable, which supports the data from Shave et al. (2010b). Recent studies have also demonstrated "positive" high-sensitivity cTnT (hs-cTnT) values after prolonged exercise in most subjects (86%–94%) (Mingels et al. 2009; Saravia et al. 2010; Scherr et al. 2011; Tian et al. 2012) but with marked heterogeneity in peak hs-cTnT (Scherr et al. 2011; Tian et al. 2012). Ongoing study is required to determine what personal, environmental, or exercise-related factors mediate this heterogeneity. On this point, it should be noted that variability in baseline cTnI is even higher than variability in peak postexercise cTnI. Whilst we observed variability in peak cTnI values, 94% of participants had their peak cTnI recorded at 3 or 6 h, which suggests some consistency in cTnI kinetics and agrees with previous data gathered after a treadmill run (Legaz-Arrese et al. 2015; Tian et al. 2012).

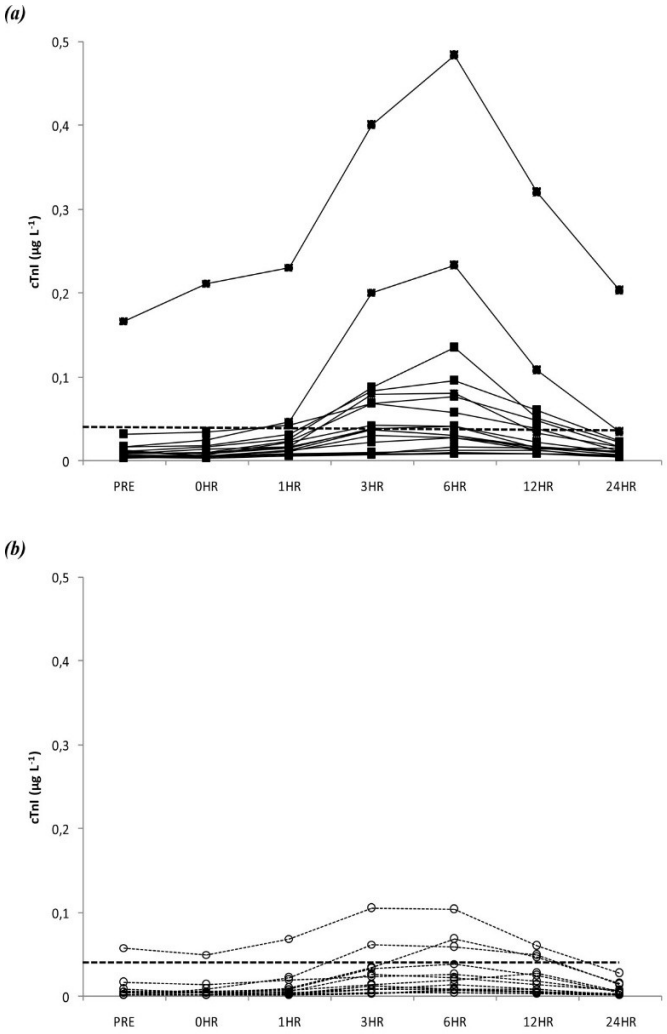
As in previous studies (Legaz-Arrese et al. 2011; Serrano-Ostáriz et al. 2011), the main factor that significantly predicted postexercise cTnI values in our study was the pre-exercise cTnI value. Across a broad range of pathologies and patient groups, baseline cTn values are repeatedly and robustly associated with an adverse cardiovascular prognosis and mortality (deFilippi et al. 2010). In healthy populations, little attention has been focused on the variability of baseline cTn values and whether this variability may have clinical significance. On this matter, our results show that athletic status may be one of the factors that determine the heterogeneity in baseline cTnI values. Further research into the factors associated with intersubject variability in baseline cTn values is required.

Certain authors have suggested that postexercise cTnI release is greater in less well-trained individuals (Fortescue et al. 2007; Mehta et al. 2012; Mingels et al. 2009; Neilan et al. 2006). However, other researchers did not observe any relationship between training level and cTnI release (Eijsvogels et al. 2015; Hubble et al. 2009; Jassal et al. 2009; Scherr et al. 2011; Serrano-Ostáriz et al. 2009). Our study demonstrated greater pre- and postexercise cTn levels in elite rowers than in amateur rowers. These data are consistent with our recent controlled endurance

training intervention (Legaz-Arrese et al. 2015) and a field-based study with marathoners (Saravia et al. 2010). Contradictions with previous studies may be related to differences in exercise regimen or training status as well as the limited number of blood samples taken during the recovery period in past work.

There has been some descriptive association between peak postexercise cTnI and mean exercise HR (Fu et al. 2009; Legaz-Arrese et al. 2011; Serrano-Ostáriz et al. 2009). Conversely, in our study, the higher absolute and relative work performed by the elite rowers during the 30-min exercise bout did not result in a greater change in cTnI during recovery when compared with the amateur rowers. This is in agreement with our recent results from a controlled endurance training intervention (Legaz-Arrese et al. 2015). Globally, current knowledge suggests that increased cTnI with exercise is associated with relative exercise intensity but not with absolute intensity or exercise performance.

Fig. 1. Individual data points for cardiac troponin I (cTnI) (g·L^{-1}) in elite (n = 18) (a) and amateur (n = 14) (b) rowers pre-exercise (PRE) as well as 0, 1, 3, 6, 12, and 24 h (0HR, 1HR, 3HR, 6HR, 12HR, and 24HR, respectively) after a 30-min maximal rowing test. The horizontal dotted line is the upper reference limit (99th percentile) at 0.04 g·L^{-1}.



We do not know the reasons behind the higher baseline cTnI levels in elite versus amateur rowers. A previous study showed that runners with detectable hs-cTnT were significantly better trained than runners in whom hs-cTnT was not detectable (Saravia et al. 2010). Furthermore, we found that a controlled endurance training intervention resulted in higher pre-exercise hscTnT values (Legaz-Arrese et al. 2015). One hypothesis is that this effect is due to successive training sessions and limited recuperation time for elite athletes. However, this seems unlikely to be a factor in this study because subjects were required to abstain from vigorous athletic activity for 48 h before the exercise test. Further, more, if the greater baseline cTnI values were a consequence of incomplete recuperation, the elite rowers ought to have had similarly increased baseline NT-proBNP levels, based on the results of this study. In a previous study, significantly higher baseline hscTnT concentrations were found in males compared with females (Mingels et al. 2009). Given that mean heart size is larger for male and elite athletes than for female and amateur athletes (Legaz-Arrese et al. 2006; Legaz Arrese et al. 2005), it is reasonable to expect different reference cTn values between these groups. Future research may wish to address this issue.

Postexercise NT-proBNP peak and kinetics in elite and amateur rowers

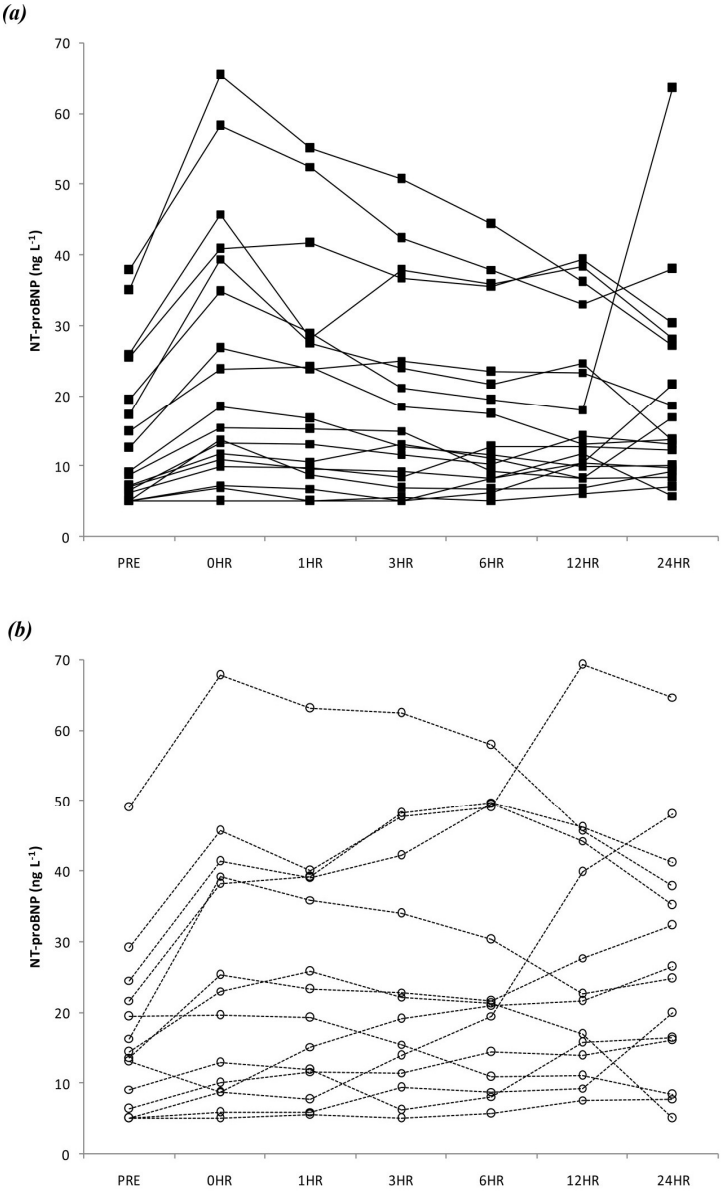
This investigation is, to our knowledge, the first study that demonstrates NT-proBNP release as a consequence of shortduration, high-intensity exercise in elite athletes. Increased NTproBNP has been reported in multiple prolonged endurance exercise studies (Legaz-Arrese et al. 2011; Neilan et al. 2006; Sahlén et al. 2008; Serrano-Ostáriz et al. 2009), and the current data extend this phenomenon to short-duration, high-intensity exercise. The observed increases are somewhat smaller than those observed in previous (ultra)endurance exercise studies (Neilan et al. 2006; Serrano-Ostáriz et al. 2009), which may not be surprising when one considers that BNP is elevated in response to volume overload and myocyte stretch (Shave et al. 2007) and that these stressors are more likely to occur during endurance exercise.

Our results demonstrate that, like cTnI values, postexercise NTproBNP values and the overall kinetics of NT-proBNP appearance exhibit a degree of heterogeneity. In agreement with the studies of Legaz-Arrese et al. (2015) and Tian et al. (2012), NT-proBNP levels in our study increased immediately after exercise and were still elevated at 24 h. The elevation in NT-proBNP at 24 h reflects an increase beyond the kinetics of NT-proBNP and its half-life (Silver et al. 2004). Other factors associated with strenuous exercise, such as a temporary reduction in kidney function and changes in cardiac function and hemodynamics, have been suggested to contribute to a sustained elevation in NT-proBNP (Tian et al. 2012), but this requires further study. It would be of great interest to analyze NT-proBNP kinetics during endurance events such as marathons, when levels are usually higher than the URL.

Our results show that although peak NT-proBNP was heterogeneous, the URL was not exceeded by any subject. Contrary to the data for cTnI, there were no apparent differences in NT-proBNP levels between elite and amateur rowers. In previous studies, the influence of training level or athletic status on NT-proBNP release has been controversial (Herrmann et al. 2003; Legaz-Arrese et al. 2011, 2015; Neilan et al. 2006; Scharhag et al. 2006; Serrano-Ostáriz et al. 2009), likely because of the inability to precisely control for several variables, such as effort duration. Our study confirms previous results showingthatbaselineNT-proBNPisakeyfactorrelated to the exercise-induced increase in NT-proBNP (Carranza-García et al. 2011; Legaz-Arrese et al. 2011, 2015; Sahlén et al. 2008; Serrano-Ostáriz et al. 2011). Interestingly, we observed greater individual variability in time to peak level for NT-proBNP than for cTnI, and consequently, previous studies may have significantly underestimated NT-

proBNP release if a single postexercise sample was taken. Future studies should be performed to determine differences in NT-proBNP kinetics among individuals after different types of exertion.

Fig. 2. Individual data points for N-terminal pro-brain natriuretic peptide (NT-proBNP) (ng·L^{-1}) in elite (n = 18) (a) and amateur (n = 14) (b) rowers pre-exercise (PRE) as well as 0, 1, 3, 6, 12, and 24 h (0HR, 1HR, 3HR, 6HR, 12HR, and 24HR, respectively) after a 30-min maximal rowing test. All values were below the URL (125 ng·L^{-1}).



Implications

We do not know whether differences between subjects in the kinetics of both biomarkers have clinical relevance. Importantly, the kinetics data for cTnI from the current study are somewhat different to those observed for cTn in acute myocardial infarction (Thygesen et al. 2012). At 24 h postexercise, all cTnI values were close to pre-exercise levels and below the URL (except the outlier). In addition, the increase in cTnI occurred in the absence of clinical signs and symptoms. This suggests that the postexercise cTnI level may reflect a physiological, rather than pathological, response to exercise stimulus. Clinicians should be aware that regardless of a patient's athletic status, cTnI but not NT-proBNP levels may exceed the URL during the first

hours of recovery after a shortduration, high-intensity exercise period in a high percentage of individuals. Since cTnI is recommended as a sensitive and specific marker for cardiac damage in the diagnosis of acute myocardial infarction, caution should be taken when interpreting postexercise cTnI levels. The results of this study are relevant for clinicians, as they could improve medical decision-making.

Strengths and limitations

Strengths of the present study include the controlled exercise regimen, matched elite and amateur rowers, serial blood sampling, and measurement of both cTnI and NT-proBNP levels. However, several limitations should be considered. First, 2 of the rowers had pre-exercise cTnI levels above the URL. Second, we analyzed associations between biomarkers and athletic status only in young male rowers. Age and sex should be studied as factors that may partially mediate the release of cardiac biomarkers with exercise (Scharhag et al. 2008; Shave et al. 2010a). The observed differences in cTnI and NT-proBNP levels between elite and amateur rowers may have resulted from differences in the level of training but could also be associated with other factors, such as genetic differences. To resolve this issue, because of the difficulty of establishing a control group with athletes, it would be interesting to observe the effect of training programs on exerciseinduced cardiac biomarker release in previously untrained subjects.

Conclusions

In conclusion, our results show that 30 min of high-intensity rowing results in elevation of both cTnI and NT-proBNP across a 24-h recovery period. Whilst increases in cTnI and NT-proBNP were observed in all rowers, the recorded peak values were highly variable, with some cTnI values above the URL. Kinetics data for cTnI were more consistent, and there does not appear to be an important role for athlete or training status in mediating exercise biomarker responses beyond the impact of potential group differences in baseline data.

Conflict of interest statement

The authors declare no conflict of interest.

Acknowledgements

We gratefully acknowledge the participation and collaboration of all elite and amateur rowers from Club Nautic Amposta (Spain). This study was supported by the DEP 2010-16767 grants from the National Plan for Research, Development and Innovation (R + D + i) MICINN.

References

Bonetti, A., Tirelli, F., Albertini, R., Monica, C., Monica, M., and Tredici, G. 1996. Serum cardiac troponin T after repeated endurance exercise events. Int. J. Sports Med. 17: 259–262. doi:10.1055/s-2007-972843. PMID:8814506.

Borg, E., and Kaijser, L. 2006. A comparison between three rating scales for perceived exertion and two different work tests. Scand. J. Med. Sci. Sports, 16: 57–69. PMID:16430682.

Carranza-García, L.E., George, K., Serrano-Ostáriz, E., Casado-Arroyo, R., Caballero-Navarro, A.L., and Legaz-Arrese, A. 2011. Cardiac biomarker response to intermittent exercise bouts. Int. J. Sports Med. 32: 327–331. doi:10. 1055/s-0030-1263138. PMID:21547864.

deFilippi, C.R., de Lemos, J.A., Christenson, R.H., Gottdiener, J.S., Kop, W.J., Zhan, M., and Seliger, S.L. 2010. Association of serial measures of cardiac troponin T using a sensitive assay

with incident heart failure and cardiovascular mortality in older adults. JAMA, 304: 2494–2502. doi:10.1001/jama.2010. 1708. PMID:21078811.

Eggers, K.M., Lagerqvist, B., Venge, P., Wallentin, L., and Lindahl, B. 2007. Persistent cardiac troponin I elevation in stabilized patients after an episode of acute coronary syndrome predicts long-term mortality. Circulation, 116: 1907–1914. PMID:17909103.

Eijsvogels, T.M., Hoogerwerf, M.D., Maessen, M.F., Seeger, J.P., George, K.P., Hopman, M.T., et al. 2015. Predictors of cardiac troponin release after a marathon. J. Sci. Med. Sport, 18: 88–92. doi:10.1016/j.jsams.2013.12.002. PMID: 24440407.

Fortescue, E.B., Shin, A.Y., Greenes, D.S., Mannix, R.C., Agarwal, S., Feldman, B.J., et al. 2007. Cardiac troponin increases among runners in the Boston Marathon. Ann. Emerg. Med. 49: 137–143. PMID:17145114.

Fu, F., Nie, J., and Tong, T.K. 2009. Serum cardiac troponin T in adolescent runners: effects of exercise intensity and duration. Int. J. Sports Med. 30: 168–172. doi:10.1055/s-0028-1104586. PMID:19199217.

Herrmann, M., Scharhag, J., Miclea, M., Urhausen, A., Herrmann, W., and Kindermann, W. 2003. Post-race kinetics of cardiac troponin T and I and N-terminal pro-brain natriuretic peptide in marathon runners. Clin. Chem. 49: 831–834. PMID:12709384.

Hubble, K.M., Fatovich, D.M., Grasko, J.M., and Vasikaran, S.D. 2009. Cardiac troponin increases among marathon runners in the Perth Marathon: the Troponin in Marathons (TRIM) study. Med. J. Aust. 190: 91–93. PMID: 19236297.

Jassal, D.S., Moffat, D., Krahn, J., Ahmadie, R., Fang, T., Eschun, G., and Sharma, S. 2009. Cardiac injury markers in non-elite marathon runners. Int. J. Sports Med. 30: 75–79. doi:10.1055/s-0028-1104572. PMID:19177312.

König, D., Schumacher, Y.O., Heinrich, L., Schmid, A., Berg, A., and Dickhuth, H.H. 2003. Myocardial stress after competitive exercise in professional road cyclists. Med. Sci. Sports Exerc. 35: 1679–1683. doi:10.1249/01.MSS. 0000089248.37173.E7. PMID:14523304.

Legaz Arrese, A., Serrano Ostáriz, E., González Carretero, M., and Lacambra Blasco, I. 2005. Echocardiography to measure fitness of elite runners. J. Am. Soc. Echocardiogr. 18: 419–426. doi:10.1016/j.echo.2005.02.002. PMID:15891751.

Legaz-Arrese, A., González-Carretero, M., and Lacambra-Blasco, I. 2006. Adaptation of left ventricular morphology to long-term training in sprint- and endurance-trained elite runners. Eur. J. Appl. Physiol. 96: 740–746. doi:10. 1007/s00421-005-0076-6. PMID:16283369.

Legaz-Arrese, A., George, K., Carranza-García, L.E., Munguía-Izquierdo, D., Moros-García, T., and Serrano-Ostáriz, E. 2011. The impact of exercise intensity on the release of cardiac biomarkers in marathon runners. Eur. J. Appl. Physiol. 111: 2961–2967. doi:10.1007/s00421-011-1922-3. PMID:21442162.

Legaz-Arrese, A., López-Laval, I., George, K., Puente-Lanzarote, J.J., Mayolas-Pi, C., Serrano-Ostáriz, E., et al. 2015. Impact of an endurance training programme on exercise-induced cardiac biomarker release. Am. J. Physiol. Heart Circ. Physiol. 308: H913–H920. doi:10.1152/ajpheart.00914.2014. PMID:25681432.

Mehta, R., Gaze, D., Mohan, S., Williams, K.L., Sprung, V., George, K., et al. 2012. Post-exercise cardiac troponin release is related to exercise training history. Int. J. Sports Med. 33: 333–337. doi:10.1055/s-0031-1301322. PMID:22377942.

Middleton, N., George, K., Whyte, G., Gaze, D., Collinson, P., and Shave, R. 2008. Cardiac troponin T release is stimulated by endurance exercise in healthy humans. J. Am. Coll. Cardiol. 52: 1813–1814. doi:10.1016/j.jacc.2008.03.069. PMID:19022162.

Mingels, A., Jacobs, L., Michielsen, E., Swaanenburg, J., Wodzig, W., and van Dieijen-Visser, M. 2009. Reference population and marathon runner sera assessed by highly sensitive cardiac troponin T and commercial cardiac troponin T and I assays. Clin. Chem. 55: 101–108. doi:10.1373/clinchem.2008. 106427. PMID:18988757.

Neilan, T.G., Januzzi, J.L., Lee-Lewandrowski, E., Ton-Nu, T.T., Yoerger, D.M., Jassal, D.S., et al. 2006. Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. Circulation, 114: 2325–2333. PMID:17101848.

Roth, H.J., Leithäuser, R.M., Doppelmayr, H., Doppelmayr, M., Finkernagel, H., von Duvillard, S.P., et al. 2007. Cardiospecificity of the 3rd generation cardiac troponin T assay during and after a 216 km ultra-endurance marathon run in Death Valley. Clin. Res. Cardiol. 96: 359–364. doi:10.1007/s00392-007-0509-9. PMID:17453141.

Sahlén, A., Winter, R., Lind, B., Jacobsen, P.H., Ståhlberg, M., Marklund, T., et al. 2008. Magnitude, reproducibility, and association with baseline cardiac function of cardiac biomarker release in long-distance runners aged ≥55 years. Am. J. Cardiol. 102: 218–222. doi:10.1016/j.amjcard.2008.03.042. PMID: 18602525.

Saravia, S.G., Knebel, F., Schroeckh, S., Ziebig, R., Lun, A., Weimann, A., et al. 2010. Cardiac troponin T release and inflammation demonstrated in marathon runners. Clin. Lab. 56: 51–58. PMID:20380359.

Scharhag, J., Urhausen, A., Schneider, G., Herrmann, M., Schumacher, K., Haschke, M., et al. 2006. Reproducibility and clinical significance of exerciseinduced increases in cardiac troponins and N-terminal pro brain natriuretic peptide in endurance athletes. Eur. J. Cardiovasc. Prev. Rehabil. 13: 388–397. doi:10.1097/01.hjr.0000219117.33038.90. PMID:16926669.

Scharhag, J., George, K., Shave, R., Urhausen, A., and Kindermann, W. 2008. Exercise-associated increases in cardiac biomarkers. Med. Sci. Sports Exerc. 40: 1408–1415. doi:10.1249/MSS.0b013e318172cf22. PMID:18614952.

Scherr, J., Braun, S., Schuster, T., Hartmann, C., Moehlenkamp, S., Wolfarth, B., et al. 2011. 72-h kinetics of high-sensitive troponin T and inflammatory markers after marathon. Med. Sci. Sports Exerc. 43: 1819–1827. doi:10.1249/MSS. 0b013e31821b12eb. PMID:21448080.

Serrano-Ostáriz, E., Legaz-Arrese, A., Terreros-Blanco, J.L., López-Ramón, M., Cremades-Arroyos, D., Carranza-García, L.E., et al. 2009. Cardiac biomarkers and exercise duration and intensity during a cycle-touring event. Clin. J. Sport Med. 19: 293–299. doi:10.1097/JSM.0b013e3181ab3c9d. PMID:19638823.

Serrano-Ostáriz, E., Terreros-Blanco, J.L., Legaz-Arrese, A., George, K., Shave, R., Bocos-Terraz, P., et al. 2011. The impact of exercise duration and intensity on the release of cardiac biomarkers. Scand. J. Med. Sci. Sports, 21: 244–249. doi:10.1111/j.1600-0838.2009.01042.x. PMID:19919634.

Shave, R., George, K.P., Atkinson, G., Hart, E., Middleton, N., Whyte, G., et al. 2007. Exercise-induced cardiac troponin T release: a meta-analysis. Med. Sci. Sports Exerc. 39: 2099–2106. PMID:18046180.

Shave, R., Baggish, A., George, K., Wood, M., Scharhag, J., Whyte, G., et al. 2010a. Exercise-induced cardiac troponin elevation: evidence, mechanisms, and implications. J. Am. Coll. Cardiol. 56: 169–176. doi:10.1016/j.jacc.2010.03.037. PMID:20620736.

Shave, R., Ross, P., Low, D., George, K., and Gaze, D. 2010b. Cardiac troponin I is released following high-intensity short-duration exercise in healthy humans. Int. J. Cardiol. 145: 337–339. doi:10.1016/j.ijcard.2009.12.001. PMID:20079546.

Silver, M.A., Maisel, A., Yancy, C.W., McCullough, P.A., Burnett, J.C., Francis, G.S., et al. 2004. BNP Consensus Panel 2004: A clinical approach for the diagnostic, prognostic, screening, treatment monitoring, and therapeutic roles of natriuretic peptides in cardiovascular diseases. Congest. Heart Fail. 10: 1–30. PMID:15604859.

Thygesen, K., Alpert, J.S., Jaffe, A.S., Simoons, M.L., Chaitman, B.R., and White, H.D. 2012. Writing group on behalf of the joint ESC/ACCF/AHA/WHF task force for the universal definition of myocardial infarction. Third universal definition of myocardial infarction. J. Am. Coll. Cardiol. 60: 1581–1598. doi:10.1016/j.gheart.2012.08.001. PMID:25689940.

Tian, Y., Nie, J., Huang, C., and George, K.P. 2012. The kinetics of highly sensitive cardiac troponin T release after prolonged treadmill exercise in adolescent and adult athletes. J. Appl. Physiol. (1985), 113: 418–425. doi:10.1152/japplphysiol.00247.2012. PMID:22653984.